Systemic and bronchial inflammation following LPS inhalation in asthmatic and healthy subjects.

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BACKGROUND: Inhaled endotoxin is known to induce airway inflammation, causing bronchial hyperreactivity. OBJECTIVE: We characterized the response to lipopolysaccharide-inhalation by measuring exhaled nitric oxide (eNO) and inflammatory mediators. PATIENTS AND METHODS: A total of 43 adult volunteers (13 asthmatics, 30 healthy controls) inhaled stepwise LPS every 30 min up to a cumulative dose of 100 microg (2.5, 10.5, 42, 45 microg). After each provocation and up to 24 h later, FEV(1) was determined; the procedure was stopped when FEV(1) declined more than 12.5%. We measured eNO, leucocytes, eosinophils, polymorphonuclear neutrophils (PMNs), C-reactive protein (CrP), lipopolysaccharide binding protein (LBP), eosinophilic cationic protein (ECP), leucotriene B4 (LTB4), thromboxane B2 (TXB2), and body temperature. RESULTS: Initial eNO values were higher in asthmatics ($P < 0.01$), but only increased in an asthmatic subgroup. Marked differences were observed in the systemic response to LPS inhalation. Significant increases were found for CrP, LBP, and PMNs. There was no correlation between FEV(1) decrease and basal eNO levels. CONCLUSIONS: Inhalation of endotoxin was followed by clinical and laboratory signs of systemic inflammation, with asthmatics responding to the challenge similar as healthy subjects. Bronchial eNO increased only temporarily in asthmatics.

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